The return to baseline by all crew members is a requirement that must be documented thoroughly. Therefore, it is necessary that the remaining Apollo and Skylab mission crews be scheduled expeditiously for postflight followup studies until the crewmen's physiologic responses have returned to preflight baseline physiologic values.

This Protocol is not approved at this time but will be implemented when approval is obtained

____ SKELETAL MUSCLE TISSUE SAMPLE

To obtain definitive intracellular quantities and distribution profiles of electrolytes and total protein in skeletal muscle.

Purpose

The purposes of this investigation are to:

1) Determine the magnitude of the total body depletion of electrolytes Na, K and total protein following exposure to the spaceflight environment.

2) Differentiate between lean body mass loss and fluid compartment loss previously postulated in the Spaceflight Adaptation Process.

3) Define the mechanism of electrolyte deficit (principally Na and K) by distinguishing between a total body deficit of electrolyte and the loss of electrolyte by muscle wasting.

4) Correlate the intracellular K^+ concentration with the total body potassium (K_E), and serum potassium as measured in previous Apollo missions.

Data Requirements

1) Skeletal Muscle Tissue sample collected according to schedule as described in Section 4.4, Table 1 of the Medical Requirements Document:

   a) One (1) sample @ F-30 days.

   b) One (1) sample @ R+0(ASAP); R+6 and R+TBD.

2) All requirements pertinent to Section 4.4 Data Requirements parts 1-3 inclusive.

Test Conditions

Skeletal Muscle Tissue sample must be acquired on specified days to insure comparability with entirety of Clinical Laboratory Medical Protocol as specified in Section 4.4.

Success Criteria

Sufficient data are to be obtained to determine deviation from accepted normal laboratory values for each parameter assayed. Samples are to be obtained at the specified frequency, processed, and delivered to the LRL in prescribed manner.

Evaluation

The single preflight values will be considered representative of the basal intracellular electrolyte state of the individuals. Postflight samples will be compared to the single preflight sample to:

1) Ascertain the magnitude of electrolyte depletion attributable to the mission exposure and adaptive response.

2) Document the return to normalcy over the postflight interval.

3) Define the trend of K_E and intracellular K recovery phases.

Background and Justification

The Clinical Laboratories have conducted extensive investigations over the previous Gemini and Apollo missions which have resulted in the formulation and partial validation of an hypothesis which describes the sequence of events leading to the adaptation of man to the spaceflight environment. While not considered as the total stress of the environment, the transition to the weighless state from earth gravity and the converse of this transition are perhaps the most significant stresses applied to the space crewman. The adaptive process is a naturally occurring event in living systems as environmental characteristics are changed; yet for the system to undergo transition to meet the demands of the new environment, a significant physiological cost is incurred. This physiological debt is classically repaid from reserves in the healthy, well-conditioned individual; however once the reserves are utilized for repayment of the incurred debt, the individual is left in a state of enhanced susceptibility to other imposed stresses. It then becomes paramount to define first the cost of the adaptation in the spaceflight environment and secondly the extent of functional reserves which remain in the adapted crewman. Without this understanding, we are unable to predict the consequences of inflight clinical stresses, such as an illness event. Such an event, should it be characterized by additional fluid and electrolyte losses, could have grave consequences.

The returning crewmen have consistently shown a total body water deficit and a significant hypokalemia. We attribute these findings to a frank diuresis which ensues upon transition to the weightless environment, resulting in a salt and water loss of sufficient magnitude to produce a secondary hyperaldosteronism. Whereas the aldosteronism is sufficient to replete the total circulatory blood volume, a persistent loss of potassium further drives the total body potassium deficit. Once the blood volume is repleted, the aldosteronism is checked and the crewmen are physiologically adapted to the new environment.

Although increased levels of plasma catecholamines, and in particular epinephrine, have been reported to lower intravascular potassium, we are unable to explain the magnitude of potassium depletion observed on the Apollo crewmen solely on the basis of this mechanism. Our interpretation of this event involves a renal tubular mechanism which promotes both Na^+ and K^+ loss in the early hours of exposure, and most likely comes about as a poorly understood consequence of the redistribution of the circulating blood volume as the null gravity is experienced. Irrespective of the cause, the end result appears to be a series of events which deplete the total body potassium. Sodium is apparently preferentially retained, either as a result of a secondary hyperaldosteronism, or perhaps as a result of a specific tubular transport process, or both. We believe the total body potassium and water deficits are consistent with a muscle mass loss principally skeletal, yet possible involving myocardial fibers as well. Confirmation of this postulate must necessarily await more critical measurements of body mass which are planned for future missions. The etiology of recorded cardiac arrhythmias is consistent with the measured levels of exchangeable potassium ion deficit. Whether or not there were other predisposing or contributory factors is not clear at this time.

In summary, we view the spaceflight-adapted individual as being in a state of compensated metabolic alkalosis with a frank hypokalemia. This adaptive process is viewed by us as a naturally occurring event upon exposure to null gravity with a physiological set of values consistent with the demands of the new environment. We are presently unable to define the magnitude of the cost of this adaptation, nor can we predict the status of functional reserve in the adapted crewman. We view the arrhythmias as a single isolated finding, in part related to the potassium deficit, yet we do not consider this finding as characteristic of the adaptive process.

The proposed muscle tissue sample when viewed in context with the entirety of the medical protocol will offer information essential to the understanding of the body electrolyte deficit. Perhaps the most important question to be resolved from this investigation is whether or not the loss of exchangeable electrolyte (principally K) results in the loss of body mass, or is the body mass loss the cause of the total body potassium deficit. A better understanding of the mechanism of the electrolyte deficit will ensue with this measurement and the specific relationship between the total body exchangeable potassium (K_E) and the intracellular concentration of electrolytes will be possible for the first time. Once these relationships are understood, the etiology of cardiac irratibility characteristic of previous missions, and the fluid, electrolyte, and body mass deficits will be conceptualized. Accordingly, understanding the underlying, contributory mechanisms operative in the metabolic control of electrolyte and fluid balance will afford definitive approaches to prophylactic or remedial courses of action to preclude adverse situations during the mission interval.